THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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OAK WILT FUNGUS ISOLATED FROM CHINESE CHESTNUT

T. W. Bretz and W. G. Long

Chalara quercina Henry, the fungus that causes oak wilt, has been isolated from five Castanea mollissima trees growing in a plantation in Missouri. The fungus was associated with a diseased condition of the trees The chestnut trees from which the fungus was isolated showed various stages of defoliation and top dying. Defoliation of fully green leaves as well as discolored leaves was taking place when the trees were observed late in August. The sapwood was discolored in some cases.

The chestnut planting is about 12 years old; there is no earlier record of serious disease in it. Chinese chestnuts in another nearby planting showed no evidence of disease. No evidence was obtained regarding possible means of transmission of the fungus to the chestnuts. Oak wilt is present in the locality. Detailed studies are planned to clarify the host relationship of the fungus.

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OAK WILT IN PENNSYLVANIA

Charles L. Fergus and Caleb L. Morris[1]

Oak wilt, caused by Chalara quercina Henry, has been found in Pennsylvania for the first time. Dead and dying trees from a mixed woods in Mifflin County, about one and one-half miles southeast of McVeytown, Pennsylvania, were reported by the owner to the Bureau of Forests and Waters, Harrisburg, Pennyslvania. Inspection of these trees later showed that three red oaks had apparently been killed several years previously since Polyporus pargamenus Fr., and P. gilvus (Schw.) Fr. were fruiting on the main stem. No isolations were attempted from these trees. One red and one black oak were dying, and exhibited the characteristic symptoms of wilt. The wilted leaves were still attached and a dark brown streaking was observed in the outer sapwood of twigs and branches. Three white oaks in the same area are suspected of being diseased. They are not yet dead, but large branches have died and numerous suckers have formed along the main stem.

Using the technique of isolation employed by Henry, et al[3], attempts were made to isolate the causal agent of the disease. Young twigs from wilting red and black oak trees, which showed the brown xylem streaking, were used. Pure cultures of a fungus were obtained. The macroscopic appearance of these cultures agreed in all details with those published by Henry[2]. No sporulation was observed until the nineteenth day after isolation. Identification of the causal agent was made more certain at this time because the fungus produced the characteristic conidiophores and endoconidia of Chalara quercina. No inoculation studies were attempted.

As far as we are aware, this is the first report of the presence of oak wilt in Pennsylvania. The distribution of the disease in Pennsylvania is not known, but it seems probable that other areas of infection are present in the State. The task of determining the distribution of oak wilt in Pennsylvania will prove difficult because of the relative inaccessibility of many oak stands and in addition, because of the tremendous acreage of forests in the State. There are approximately 15 million acres of wooded land in Pennsylvania and about one-half of the trees are oak.

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[1]Forester, Division of Research, Department of Forests and Waters, Commonwealth of Pennsylvania, Harrisburg, Pennsylvania.

[2]Henry, B. W. Chalara quercina n. sp., the cause of oak wilt. Phytopath. 34: 631-635, 1944.

[3]Henry, B. W., C. S. Moses, C. Audrey Richards, and A. J. Riker. Oak wilt: its significance, symptoms, and cause. Phytopath. 34: 636-647. 1944.

AN EPIPHYTOTIC OF WHITE OAK ANTHRACNOSE IN CENTRAL PENNSYLVANIA

Charles L. Fergus

Anthracnose of Quercus alba L. caused by Gnomonia veneta (Sacc. and Speg.) Klebahn has been extremely severe this year in central Pennsylvania. Many white oaks on the campus of The Pennsylvania State College, Centre County, State College, Pennsylvania, exhibited the characteristic lesions of the disease on the leaves and some twig dieback as early as May 22, 1950. White oaks on the campus have been diseased for a number of years, but with the exception of 1944, the incidence has been relatively low. Overholts (4) for Gnomonia veneta recorded: "In 1944 there was a rather severe epidemic of this fungus on Quercus alba at State College, Pa. Other species were not affected."

Because of the severity of the disease this year the writer made observations on the number of diseased trees and on the amount of infection on 58 white oaks in a limited area on the campus. This area is about one acre in extent and is maintained as a park area, although the white oaks originally were part of a natural stand of timber. Some trees have over a period of years been removed, so that now there is a much larger space between trees than would ordinarily be found under forest conditions. The natural undergrowth has been removed and replaced with a turf of thick grass. The area is frequently mowed, and during fall the leaves are raked and removed. The trees therefore are subject to an artificial environment, quite dissimilar to forest conditions. White oaks under such conditions, on park areas and on lawns, have suffered much more from anthracnose than white oaks under forest conditions.

Every white oak in the area was examined. The amount of infected foliage was also estimated. Table 1 summarizes the observations.

Table 1. Summary of the number of diseased white oaks and the approximate amount of the foliage infected by Gnomonia veneta.

Amount of anthracnose	Number of trees
None	5
Slight[a]	15
One-fourth	2
One-third	14
One-half	14
Entire tree	8
Total trees examined	58

[a]Slight indicates any amount of infection less than 25 percent of the leaves of a tree.

In the cases where only a part of the tree was infected, the diseased leaves were without exception on the basal branches. Edgerton (2) regarded the conidia of the Myxosporium stage on the twigs as the most important primary inoculum, but undoubtedly ascospores of the perfect stage produced on fallen overwintered leaves also serve to establish the disease. Although the area examined is usually raked several times during the fall, many leaves or diseased leaf fragments resulting from the shattering of the brittle necrotic tissue could be missed, allowing the overwintering stage to develop.

No specific data are available for the severity of anthracnose on white oaks under forest conditions, but from casual observations made by the writer, there is a considerable amount of anthracnose of white oak in forests, although the disease seems less severe than in the area examined on the campus. Members of the Plant Pathology Extension Department have reported that anthracnose is abundant in many areas throughout the State this year.

Meteorological data for State College, Pennsylvania (1) were examined in an attempt to correlate weather conditions and the recorded epiphytotics of 1944 and 1950. The data are presented in Table 2.

The weather conditions which prevailed in both 1944 and 1950 and which appear to have determined the occurrence of severe anthracnose are: (a) an open winter with temperatures of

Table 2. Summary of weather conditions in State College, Pa. for the past seven years.

Weather	1944	1945	1946	1947	1948	1949	1950
Sum of winter[a] temperatures	88.4	75.2	83.9	90.7	79.2	101.5	98.8
March temperature °F	33.9	49.2	47.4	32.0	40.5	38.9	31.7
Departure from 68-year normal	-2.0	+13.3	+11.3	-4.3	+4.2	+2.6	-4.6
April temperature °F	45.6	53.1	49.6	49.2	51.1	48.7	43.6
Departure from 68-year normal	-1.8	+5.8	+2.2	+1.7	+3.6	+1.2	-3.9
March snow (in inches)	17.9	1.2	0.5	17.2	5.1	5.2	23.4
Precipitation in April (inches)	4.05	4.38	0.63	3.20	6.59	2.75	1.95
Precipitation in May (inches)	7.83	6.09	7.57	4.42	4.54	3.27	4.10
Precipitation accumulation departure through May	+1.1	+3.3	-1.2	-4.3	+1.8	-0.1	+2.1
Percent hours sunshine possible	37.8	49.1	52.0	41.8	40.0	54.1	38.6

[a]Sum of mean temperatures of December, January, and February in degrees Fahrenheit. Normal for 68 years is 83.4.

December, January and February higher than normal, (b) a delayed spring with deep snow cover in March and temperatures of March and April lower than normal, (c) moderate precipitation recorded for a large number of days (23 days in April, 1950 and 15 days in May, 1950), and (d) low percentage of effective sunshine hours possible (preventing rapid evaporation of moisture).

It is interesting to note that some of the above weather conditions, believed to be important in the development of white oak anthracnose, are similar to those found by Coe and Wagener (2) to be important in the development of ash anthracnose caused by Gloeosporium aridum Ell. & Holw. These workers state that: "A delayed spring, marked by prolonged rains and cool temperatures, no doubt provided very favorable conditions for the development and spread of this disease in 1948."

Further observations will be made during the coming years to determine the accuracy of the opinion that the weather conditions stated above are requisite for the occurrence of an epiphytotic of white oak anthracnose.

Literature Cited

1. Anonymous. Meteorological data for State College, Pa. Meteorology Dept. Penn. State College. (Unpublished).
2. Coe, Donald M., and Willis W. Wagener. Ash anthracnose appears in California. Plant Dis. Rptr. 33: 232. 1949.

3. Edgerton, C. W. The physiology and development of some anthracnoses. Bot. Gaz. 45: 367-408. 1908.
4. Overholts, L. O. Studies in the fungi. (Unpublished).

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THE CACHEXIA DISEASE OF ORLANDO TANGELO

J. F. L. Childs

The Orlando tangelo, a hybrid citrus variety, is the result of a cross between Bowen grapefruit and Dancy tangerine, that was made by W. T. Swingle about 1900 at Eustis, Florida. The hybrid was first named the Lake tangelo, and was described in 1931 by Swingle, Robinson, and Savage (3). In conversation the name Lake frequently was confused with "late"; actually it is a very early tangelo; hence the fruit was given the name Orlando (4). While the fruit has been favorably regarded for many years, few were grown commercially. Within recent years a demand has arisen for fancy fruits suitable for the express shipment trade, and as a result Orlando tangelos are being more widely planted. The present recorded planting is about 33,000 trees, of which 18,000 are in bearing, or approximately 275 acres on the basis of 65 trees per acre. These figures undoubtedly fall far short of the actual acreage. Competent observers place the acreage in bearing at not less than 500.

A disease affecting the Orlando tangelo was first called to our attention in 1945 in a planting of this variety on Cleopatra rootstock near Clearwater, Florida. Subsequently the disease was found near Lake Butler, Apopka, Groveland, Tavares, Avon Park, and Sarasota in Florida. Affected Orlando trees were found on the following rootstocks: sour orange, sweet orange, Rough lemon, Rusk citrange, and Cleopatra mandarin. It has also been found on Orlando scions that were topworked on old grapefruit trees. This trouble has been referred to as the "Orlando tangelo disease," but "cachexia" (kakos, bad, and hexis, condition), meaning malnutrition and wasting due to some chronic consititutional affection, more accurately describes the disease.

The number of trees that are diseased is unknown, but in the plantings observed to date it varies from less than 1 percent to over 60 percent. Many older plantings appear to be practically free of the disease, but the more recent plantings observed average around 25 percent sick trees. As a result of that situation, many growers are reluctant to plant this excellent variety of citrus.

Symptoms of the Disease

Vein Chlorosis: A conspicuous symptom and perhaps the first one noted by the grower is the appearance of chlorotic leaves. Yellowing is most pronounced along the midrib region of the leaf and is typical of starvation symptoms (Fig. 1). Chlorosis may largely disappear after fertilizer application, but returns within a short time on the affected trees. Meanwhile, the foliage of adjacent unaffected trees remains green from one application of fertilizer to another. More or less defoliation may accompany the chlorotic condition.

Stunting: Affected trees can be detected by their conspicuously stunted appearance, without regard to color of the foliage (Figs. 2 and 3). Stunting usually becomes apparent within the second or third year from the time the trees are set out and becomes more noticeable as time goes on.

Cankers: Another conspicuous symptom is the appearance of cankers on the trunk and occasionally on the branches of affected trees. These appear most frequently just above the union of the Orlando and its rootstock (Fig. 4). Cankers have never been observed to progress any distance below the bud union, although they may advance upward and also around the circumference of the trunk in such a manner as partially to girdle it. Apparently the transfer of nutrients is impeded even before the bark lesions appear, because vein chlorosis is not confined to trees on which cankers have appeared.

Phloem Discoloration: A fourth symptom may be noted even before the appearance of either the stunted condition or the cankers, and that is the appearance of a dark brown discoloration in the phloem region of the scion variety. This discoloration is readily seen in tangential (Fig. 5) or radial sections (Fig. 6) and is sharply delimited by the line of the union between the scion and the rootstock. The distance to which the discoloration may extend above the bud union is unknown but has been traced more than 18 inches. The bark of small branches in the tops of affected trees has not been found to be discolored. No discoloration has been seen to take place in the bark of any of the several rootstocks.

Stem-Pitting: A fifth symptom, and perhaps the one least likely to be noticed by the grower, is stem-pitting, or, more properly, wood-pitting. When the bark is peeled off at the bud union on affected Orlando tangelo trees it is found that the surface of the exposed wood of the scion is indented or pitted in a very uneven manner. The cambial surface of the removed bark is marked by bumps and projections that coincide with and fit into the depressions in the wood (Fig. 6). Below the bud union the exposed wood and the cambial surface of the bark is smooth and normal

Figure 1--Leaves of diseased Orlando tangelo, showing yellow midrib and veins.
Figure 2--Healthy Orlando on rough lemon rootstock, five years after setting out.
Figure 3--Diseased Orlando in the same planting as the tree shown in Figure 2.
Figure 4--Trunk canker of diseased Orlando on Rough lemon root.
Figure 5--Bark scraped to expose the discolored region in the phloem. Note how the boundary between the scion and the rootstock is followed.
Figure 6--Strips of bark cut across the bud union from diseased Orlando on Rough lemon root. Rough upper portion is Orlando bark. Note discoloration showing in the edge of the piece on the left.

to all appearances. Occasionally affected trees produce a number of fruits with very bumpy rind. The significance of this character and its relation to the disease is unknown.

Causal Relationships

Incompatibility: There seems to be no evidence of any incompatibility between the various species of citrus used as rootstocks and the Orlando tangelo scion that can account for the development of the above symptoms. Several Thornton tangelos on Rough lemon root have been found showing the same symptoms of stunted condition and discolored bark. Beyond the observation that the disorder appears to be similar to the "Orlando tangelo disease", little is known about the disease in the Thornton.

Boron Deficiency: Certain aspects of the disease, particularly the chlorotic foliage and disorganized bark tissues, suggest the possibility of boron deficiency, but it has not been possible to demonstrate any such relation. In one experiment boron was applied to affected trees five years old in amounts of 2 and 4 ounces in each of three applications at intervals of two months. Following these applications, evidence of boron toxicity appeared, but there was no change in the chlorotic appearance of the foliage. Leaf samples from a 99 percent healthy grove of Orlando trees on Rusk citrange root were analyzed by Dr. P. F. Smith of this station, who found 37 and 42 p.p.m. of boron in the last flush of growth on two trees and 182 and 182 p.p.m. in samples from the previous flush. In a nearby grove of Orlando tangelo on Rusk citrange where 46 percent of the trees were affected, two healthy trees showed 32 and 140 p.p.m. in leaves of the last flush and 107 and 125 p.p.m. in leaves of the previous flush. In the latter grove two sick trees showed 32 and 37 p.p.m. in leaves of the last flush and 140 and 172 p/p.m. in leaves of the previous flush. In a planting of Orlando on Rough lemon the boron content of leaf samples from the last

flush on four healthy trees was 38, 38, 45, and 38 p.p.m. Similar samples from four diseased trees showed 28, 35, 33, and 30 p.p.m. of boron. In this grove alternate rows of trees were sprayed with borax at the rate of 1 pound per 100 gallons. There was no change in health or greenness of foliage of either the diseased or healthy trees. None of the above results suggest that boron shortage has any relation to the disease.

Fungus or Bacterial Infection: Numerous attempts have been made to isolate a causal organism from the margins of trunk cankers. In the main the cultures were sterile, although a small portion proved to be contaminated with a species of Fusarium or with unidentified bacteria. Typical were a series of isolations made at the Lake Butler grove, in which 86 percent proved sterile, 5.5 percent yielded Fusarium sp. and 8.5 percent yielded unidentified bacteria. Results such as these have been regarded as evidence against the causal organisms being of a fungal or bacterial nature, because of the high proportion of sterile cultures. If a fungus or a bacterium were involved there should have been relatively few sterile cultures, even though the pathogen were not recovered, owing to the rapidity with which saprophytes follow invasion by a pathogen under subtropical conditions. Microscopic examination of stained freehand sections cut from the margins of cankers revealed no evidence of microorganisms. The fact that these cankers very seldom exude any gum, even under conditions favorable for copious gumming from Phytophthora and Diplodia lesions, may be further evidence against the presence of pathogenic microorganisms.

Virus Relations: In one Orlando tangelo grove leaf patterns similar to those associated with psorosis were found on three occasions. A count was made on one occasion when leaf symptoms were particularly striking. Of 64 trees that showed discoloration of the phloem 28, or 44 percent, showed leaf patterns. Some trees showed oak-leaf patterns of the type illustrated by Klotz and Fawcett (1). It is not known whether such patterns are peculiar solely to citrus trees affected with psorosis or are a symptom expressed by citrus when infected with any one of a number of viruses. Recently, specimens of bark from the bud union region of both diseased and healthy Orlando trees were killed and fixed and sent to Dr. Henry Schneider at the California Citrus Experiment Station for study. Without going into a detailed account of his findings as to the changes in structure in the diseased bark specimens as compared with healthy specimens, it is sufficient for the purposes of this paper to state that in Dr. Schneider's opinion the trouble is of virus origin.

Oberholzer, Mathews, and Stieme (2) have described an insect-transmitted wood-pitting virus disease of grapefruit trees in South Africa in which the stunted habit of growth of the affected trees is much like that found in the Orlando tangelo disorder. Recently Dr. A. P. D. McClean at Pretoria, South Africa, sent the writer photographs of bark, stems, and leaves of citrus trees affected with stem-pitting virus. Included was a photograph of the cambial face of a strip of bark from a diseased tree of Bailedge Early orange, and the ridges and bumps shown are rather similar to those found in the bark of diseased Orlando tangelos in Florida.

Bud Transmission Experiments: Experiments were started in 1947 to determine whether the disease is transmissible through infected buds. For that purpose, 270 one-year-old Rough lemon seedlings were set out in lots of thirty each. The seedlings were budded in place as follows: (a) buds from diseased Orlando tangelo trees, as determined by their stunted habit of growth and by the phloem discoloration test, were inserted into three lots of seedlings. The buds used in each of the three lots came from several different groves; (b) buds from healthy Orlando trees, as determined by vigorous habit of growth and by negative results in the phloem discoloration test, were inserted into six lots of seedlings. Buds used in two of these lots came from healthy trees in the same groves from which diseased buds were obtained, buds for two other lots came from a grove of Orlando trees that were 99 percent healthy, and buds for the remaining two lots were obtained from two healthy Orlando trees in the U. S. D. A. collection of citrus varieties. The Rough lemon seedlings were budded in the fall of 1947 and cut back to the bud in the following spring. Records were kept of the bud source for every budded tree, but many of the diseased parent trees have since been destroyed by their owners.

One lot of trees carrying healthy Orlando buds was budded at the same time with psorosis-infected buds from a Valencia orange tree affected with that disease. In each case the virus-infected bud was inserted in the Rough lemon stock below the Orlando bud. In 1947 one lot of trees carrying healthy Orlando buds was rebudded with buds from a Dancy tangerine tree infected with the concave gum form of psorosis. Early in 1950 buds from diseased Orlando trees were inserted in one block of healthy Orlando budded trees. In the same year twelve Persian lime trees budded on sour orange were planted in 14-inch pots in the greenhouse. Into six of these trees buds from diseased Orlando trees were inserted, while for the other six, buds from healthy trees were used. In this transmission test two buds were placed in each tree.

To date, two years after the appearance of the first growth on the Orlando buds, no leaf symptoms definitely assignable to virus origin have appeared on any trees, with the exception of

those in which psorosis buds were inserted as described earlier. By the spring of 1949, one and a half years from budding, 24 of the 30 trees displayed typical leaf symptoms of psorosis. Up to the present time (June 1950) only a few of the larger trees have been examined for discoloration of the phloem region of the bark and none has given a positive test. No leaf symptoms have appeared on the lime trees, although McClean found rapid expression of symptoms of South African stem-pitting virus on this variety.

Discussion

In the absence of conclusive data from transmission experiments, it is impossible to state that this disorder of Orlando tangelos is of virus origin. Certainly it has many attributes of a virus disease, namely, leaf symptoms (although of sporadic appearance) and disorganized areas in the phloem, together with stem-pitting. So far as can be determined, there is no transmission from tree to tree in the field. Healthy trees appear to remain so, and diseased trees have not been observed to recover, except temporarily following applications of fertilizer. The existence of trees that may be symptomless carriers is a possibility suggested by the following observation. The worst example of the disease seen so far appeared in a small planting of grapefruit that had been cut back and topworked with Orlando scions. Nearly every scion examined showed phloem discoloration and many showed cankers, although the buds came from a grove in which very few trees were affected. To date the type of symptoms associated with "Orlando tangelo disease" have not been found on grapefruit trees in Florida.

The evidence gathered to date is against the possibility of a fungus or bacterial pathogen in this disorder, and there is good evidence against the possibility of boron deficiency. Rootstock-scion incompatibility seems to be out of the question, since healthy and diseased trees are found on the same rootstock varieties and under the same conditions of growth.

Recommendations for Control: Since there is some indication that a virus may possibly cause the cachexia disease of Orlando tangelo, it is recommended that buds for propagation purposes be obtained only from healthy trees of seven or more years of age, if possible. The disease usually shows up within three years of the time a tree is set out, so that a seven-year-old tree of healthy appearance that shows a negative test for phloem discoloration and is free of wood-pitting should be a safe source of budwood.

Literature Cited

1. Klotz, L. J., and H. S. Fawcett. Color Handbook of Citrus Diseases. pl. 22. Univ. of Calif. Press, Berkeley, Calif. 1941.
2. Oberholzer, P. C. J., I. Mathews, and S. F. Stieme. The decline of grapefruit trees in South Africa: A preliminary report on so-called "Stem-Pitting." Dept. of Agric., Union of South Africa, Agric. Research Inst. Series No. 19, Science Bull. 297. 1949.
3. Swingle, W. T., T. R. Robinson, and E. M. Savage. New citrus hybrids. U. S. Dept. of Agric. Circ. No. 181, p. 6. 1931.
4. Traub, Hamilton P. Improvement of subtropical fruit crops. U. S. Dept. of Agric. Yearbook. pp. 766-767. 1937.

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SOME NOTES ON APPLE DISEASE CONTROL IN NEW HAMPSHIRE

M. C. Richards

Apple Scab Control with Sulfur-NuGreen Combinations

It is noted that Poulos and Heuberger (Plant Dis. Reptr. 34: 239. 1950), and Stoddard (Abs. Phytopath. 40: 27. 1950) report better scab (Venturia inaequalis) control with sulfur-NuGreen combinations than with sulfur alone. Our experience in New Hampshire during the past two years has shown that a thorough job of ptorection must be accomplished with the combination, or a more serious scab infection will occur than with sulfur alone, as illustrated by the data below.

Foliage scab, McIntosh variety at Durham test plots

Treatments	Percent foliage June 16	Scab 1950 July 13
Kolospray (Sulfur) 5-100	7.6	8.3
Kolospray-NuGreen 5-5-100	33.0	64.0
Phygon XL 1/2-100	0.0	.3
Phygon-NuGreen 1/2-5-100	4.0	1.0
Puratized Agr. Spray 1 pt.-100	.6	.3
Puratized Agr. Spray-NuGreen 1 pt.-5-100	0.0	0.0
Unsprayed Check Trees	51.0	88.0

Spray applications were made at pink, early-bloom, calyx, and first-cover. Fungicide applications were purposely delayed in the early-bloom and calyx sprays to permit some infection so that these materials and others could be checked for eradicative properties. NuGreen-treated leaves seem to be much more susceptible to scab; therefore, it is believed that in the hands of a grower a better protectant than sulfur or a material with eradicant properties should be used in combination with NuGreen.

Cedar and Quince Rust on Apples

The period May 29 to June 1 was very favorable for spread of cedar and quince rust (Gymnosporangium juniperi-virginianae, G. clavipes) to apples. About 1 percent of the fruits on the sulfur-sprayed trees on the Delicious variety were affected with quince rust, while ten to fifty cedar rust lesions could be counted on the McIntosh leaves. Fully developed pycnia were produced but only rudimentary aecia were formed on this variety.

Phygon gave better control of cedar rust on the apple leaves than Ferbam.

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† PRELIMINARY REPORT ON THE CADANG-CADANG DISEASE AND ON SOIL DEFICIENCY TROUBLES OF COCONUTS IN THE PHILIPPINES

Otto A. Reinking[1]

The apparently infectious disease called cadang-cadang, as it occurs in the Bicol region of the Philippine Islands, already appears to be a limiting factor in coconut culture in certain areas and must be taken seriously. The cause is as yet not definitely determined, although Celino (3), in preliminary studies which need verification, apparently transmitted a virus from diseased parts of severely affected coconut trees to healthy coconut seedlings.

In this report the term cadang-cadang will be used only for the so-named disease now so prevalent in and apparently confined to the Bicol region. The other troubles investigated, which also cause serious reduction in yield, will for the present be referred to as soil deficiency disorders.

The preliminary conclusions arrived at in this discussion were based on three thorough surveys of affected regions. From May 22 through May 26, 1950, an intensive study was made of the disease situation in the Bicol region. Severely affected cadang-cadang areas were studied in the provinces of Camarines Norte, Camarines Sur, Albay, and Sorsogon. Detailed studies were made in the San Miguel Estate on the Island of that name in Albay Province. In Camarines Norte, the presence of the disease was merely verified near the airfield at Daet; in Sorsogon also I merely verified presence on the way to Pilar. The following members of the Bureau of Plant Industry were present during the main part of the inspections: Drs. F. Q. Otanes, T. G. Fajardo, Dionisio Aquino, and Mr. Jose L. Morales. Mr. P. B. Marcelo, Provincial Agricultural Supervisor of Camarines Sur, was of exceptional assistance in arranging interviews with growers and providing transportation.

To study the deficiency trouble a survey was made in the Candelaria, Quezon, region from May 31 through June 2. Arrangements were made through Mr. Virgil F. Murray, Provincial Manager of the Peter Paul Corporation at Candelaria, and Mr. H. Dean Hollis of the Philippine Refining Company, Inc., of Manila. Mr. Murray had reported a diseased condition in the Candelaria region in March 1950. Areas in which yields had been reduced to a point of uneconomic production were inspected, and the cause was tentatively designated as a deficiency of plant nutrients due to various factors. The great difference in symptoms and behavior between the deficiency troubles as they appeared in the Candelaria region and the cadang-cadang disease in the Bicol region was clearly evident from these studies.

To clarify certain questionable points in regard to the cadang-cadang disease, a second survey was made on San Miguel Island, Albay, from June 6 through June 9, 1950, in company with Dr. Fajardo and Mr. Murray. Arrangements for the visit to the Island Estate were made through the owner, Mr. Santiago Caucayco of Manila, and his manager, Mr. Julio San Jose.

CADANG-CADANG DISEASE

Cadang-cadang disease was first reported as appearing on the Island of San Miguel, Albay, in 1926 (2). In 1937, Ocfemia (6) discussed its probable nature. A preliminary account of the disease as it occurred on the San Miguel Estate was given by Celino (2). In these reports, the symptoms as now present were accurately described. Celino reported experimental transmission (3), and gave a general review of the disease as it occurred in the Philippines in 1947 (4).

Symptoms

General Appearance of Badly Affected Trees: Diseased trees typically show a general yellow-bronzing, especially on the older lower leaves; not a distinct yellowing of the foliage. Young leaves in the center of the crown may remain green except in very advanced cases when they may become somewhat yellowed. In advanced cases small thin crowns of shortened, rather upright leaves are all that is left of leaf production; in these cases all leaves may be yellow-bronze. Few browned, dead older leaves remain hanging from the crown around the trunk of badly diseased trees; apparently, old dead leaves drop off much more freely than on trees affected with the deficiency trouble. Occasionally, central green leaves fail to unfold, leaving an upright tuft in the top of the tree that appears cut off at right angles. The growing point and leaves eventually die, leaving a tuft of dried leaves on top. Death may take place in five to six years after infection (2).

No typical symptoms were observed on the floral parts of recently infected trees. In older infections the floral parts become stunted. Production of nuts and flowers is gradually reduced as

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the disease progresses and ceases in advanced cases.

The taste of milk from nuts of infected trees was normal. Buds of diseased trees tasted sweet except for a few instances of a slightly bitter flavor.

Leaf scars on the trunks of badly affected trees, just below the oldest living leaves, are very close together, indicating poor slow growth during the last two years or so. In such cases, the trunk may taper at this point, and a stunting in growth results.

Trunks of diseased trees were apparently normal in cross-section, with no discoloration of the vascular bundles that might indicate a vascular disease. Growing roots of diseased trees appeared healthy, with no discoloration in the stele. On badly diseased trees many roots were dead, which might be regarded as a normal condition for a weakened tree.

Typical Symptoms on Leaflets, Petiole, and Mid-rib of Badly Affected Trees: A yellow mottling that may be observed from the ground when looking through the leaflets into the sun is the most evident symptom on leaflets; it generally appears in spots or blotches. Blotches, by running together, may develop into intermittent or continuous yellow streaks 1 to 2 mm. wide, along both sides of the main veins of the leaflets. The yellow mottling is more pronounced on leaves of yellow coconut varieties as compared with the green nut varieties.

Water-soaked spots and blotches, 1 to 3 mm. wide, are characteristically present on the under surface of badly affected leaflets, in association with the yellow mottling or separate. These watery spots may occur on the second or third unfolded leaves without the yellow mottling. Often on younger leaflets, even on the third youngest unfolded leaf, a whole series of water-soaked spots may run along both sides of the mid-rib or be scattered over the surface. Such symptoms are very similar to those of a bacterial infection; however, microscopic examination failed to show any evidence of either bacteria or fungi.

Occasionally, leaflets were observed with definite irregular darker blotching covering a third to a half of each side of the mid-rib. This symptom was not general and may have been due to a combination of causes.

In severe cases, younger leaflets may be much narrowed, tough, and more brittle and greener than normal leaflets; they may be malformed, broken in the middle, and crinkled on the edge. Such leaflets may show the watery spots without the yellow mottling. These symptoms are typical virus disease symptoms.

Water-soaked spots, blotches, and streaks may be present petioles and mid-ribs of the leaves The tips of older leaves generally show more yellowing owing to more intense mottling of the leaflets. On these older leaves drying, browning, and death of the leaflets may occur.

Importance

The disease is reported as most severe in Albay and Sorsogon Provinces. There are areas in both of these Provinces where 90 to 100 percent infection is present. In the vicinity of Logaspi, Albay, severe infection up to 100 percent is common. In the Daraga, Albay, area up to 90 percent infection is reported. In the Nato area of Camarines Sur, areas with 90 to 100 percent infection were observed. Similar figures could be presented for other sections and areas in Sorsogon and in the Daet region of Camarines Norte.

One clear-cut example, among others, of destruction due mainly to the cadang-cadang disease is presented on the San Miguel estate in Albay Province. Land for this estate was cleared in 1920 and the main part planted by 1924. Of the 260,000 bearing trees at the peak of the planting, 116,-000 were standing in 1950. It was reported that the main reduction was caused by cadang-cadang disease, although the typhoon of December 25, 1947 undoubtedly accounted for some loss. Our observations would indicate that the disease was chiefly responsible. Many of the trees now standing are diseased and producing below-normal crops. Production in 1947 was 260 tons of copra, in a 12-month period in 1949-50 it was 55 tons.

Observations on San Miguel Island, Albay Province

The detailed study of the disease as it is now present on San Miguel Island, Albay, brought out many interesting factors. These investigations were of extreme value because there existed earlier records of prevalence and symptoms with which to make comparisons on the spread and nature of the trouble. These records date back to observations made by Ocfemia in 1930 (6) and by Celino in 1947 (2, 4).

In 1930, according to Ocfemia, the most severe infection amounted to about 25 percent. In 1947 Celino (2) reported up to 85 to 90 percent infection in these same areas. He further reported up to 85 to 90 percent disease in Sections I, II, III, and IV of the San Miguel Estate. He noted,

however, that Sections V and VI showed only a mild form with about 2 to 3 percent infection.

Our inspection in May and June, 1950, indicated further spread and increased severity. In large areas of Sections I to IV disease was present in 80 to 100 percent of the trees and nut production was seriously reduced. Spots of better trees, however, with only 20 or 50 percent disease, were present in parts of each section. This was particularly true of lower areas in small ravines in Section IV; not over 20 percent disease was noted in these locations and nut production was good. Spread from diseased trees marked in 1948 apparently was slower in these healthier areas; which seemed to be more protected from wind damage and possibly were not so seriously affected by the typhoon of December 25, 1947. Perhaps growing conditions were better. Such areas might show a difference in soil types if detailed soil surveys were made. In Sections V and VI, in which Celino (2) had noted only 2 to 3 percent infection, large areas still showed not over 20 percent in 1950. In some of the southern parts of Section V infection reached 80 to 100 percent. In large areas of this section, nearer the coast on the west side of the Island, not over 20 percent was observed; these areas again appeared to have better wind protection and the soil type, of course, might also be different. In Section VI the disease was more general, with spots showing only 50 to 60 percent, but other areas with 80 to 100 percent infection. Evidently, disease spread in Section VI had been rather serious.

It is evident, therefore, that the disease had spread from the time of Celino's report in 1947 (2) to May and June of 1950. The production data given above would indicate a severe reduction in yield to have taken place, which must, in part at least, be assigned to the disease.

Trees planted on San Miguel Island during the occupation, in 1942-1943, as yet showed no signs of cadang-cadang. A rather heavy attack of Pestalotia leaf spot was observed. These seven- to eight-year-old trees were not in bearing as yet. Older bearing trees, 15 to 20 years old, adjoining were badly affected by cadang-cadang. Volunteer coconut trees, three to four years old, growing among badly infected trees in a 1924 planting in Section III, also showed no infection. Here again, young trees that as yet had produced no flowers showed no sign of infection even though growing under severely infected trees. All these young trees should be carefully watched in the future for disease development.

Trees reported to have been planted in 1932-1934, therefore some 16 to 18 years old, were among the youngest trees in bearing that were observed. They were severely affected with cadang-cadang disease. Infection on these trees was noted to be present even on the second and third unfolding young leaves.

Elsewhere in the Bicol Provinces occurrence was in all respects similar to that reported for San Miguel Island. The symptoms were always the same. In no cases were young trees before flowering noted to have the disease.

Discussion

The fact that young trees, before bloom and nut production, were in all cases free from disease would indicate either that infection (if this is an infectious disease) takes place through the floral parts or that the disease if present is latent and does not show symptoms in such young trees.

Trees marked in 1948, on San Miguel Island, and treated for disease by burning the lower leaves were in every case in the last stages of disease in 1950, indicating that badly affected trees do not recover.

The presence of isolated very productive trees in areas of 90 to 100 percent infection on San Miguel Island, and of similar examples found in all of the other severely diseased areas in the Bicol region, would indicate that soil deficiency is not the cause of the disease.

The so-called infectious cadang-cadang disease has distinct and characteristic symptoms that were always present in affected areas studied in the Bicol Provinces. The observations reported here would indicate that the disease is infectious. It has spread badly in the entire Bicol region and is present in severe form in large areas.

Microscopic examination of the water-soaked spots and blotches on affected leaves failed to show the presence of bacteria and fungi. Further study is needed, but these preliminary investigations tend to substantiate Celino's virus findings (3).

Of the many topics for research suggested in the following list many are already under way. Detailed studies on cause and method of transmission should be conducted to verify and amplify Celino's investigations (3); the possible association of bacteria or fungi should be checked by isolation studies; possibility of infection through the inflorescence needs thorough study; the role of insects as vectors should be determined, and especially insects on floral parts should be subjected to detailed study; further investigations should be made on possible infection of the vascular

system and the roots (5); selection of potentially resistant varieties and breeding for resistance should be continued (1). Fertilizer tests should be amplified.

A detailed study of field spread and possible recovery should be initiated. Charts with data showing the status of the disease in key areas over a period of years are essential as a basis for control. Plots in many type areas of each affected Province should be mapped in detail and observed monthly for rate of spread, increase in severity, possible recovery, etc. In some localities the disease appears to be spreading much more rapidly than in others. Accurate long-time records showing exact field spread in all type situations must be obtained.

Accurate soil surveys and analyses of surface and subsoil might help to explain possible differences in rate of spread under a variety of conditions.

A careful check should be made of yellowed coconuts in all areas of the Philippine Islands. The extent of spread of cadang-cadang must be definitely established.

An excellent new laboratory and area for experimental plots has been established at the Guinobatan Agricultural Experiment Station in Albay Province. Facilities are available also at the Bureau of Plant Industry Station in Manila.

SOIL DEFICIENCY TROUBLES OF COCONUTS

The following account is based on preliminary studies made in the Candelaria region in Quezon Province, from May 31 through June 2, with Mr. Virgil F. Murray as reported above. Until detailed investigations have been made the trouble in the Candelaria area has been designated as due to a possible soil deficiency brought about by various factors.

Symptoms

The main symptoms are a general yellowing of the older living leaves and a more than normal number of browned dead leaves hanging from the top of the tree around the trunk. Older leaves turn brown and die prematurely, apparently because of lowered vitality due to soil depletion. In moderately affected trees the central leaves may be a normal green, but in poorer trees even the top youngest leaves may be yellowed. The top leaves may be shorter and smaller than normal leaves.

The leaves of trees weakened by the apparent deficiency show a definite yellowing, not at all resembling the yellow-bronzing of cadang-cadang. There is none of the yellow mottling and watery spotting typical of cadang-cadang on leaflets of trees affected by the deficiency trouble. On cadang-cadang affected trees old dead leaves fall off after early maturity; in the case of the deficiency trouble they hang on the tree longer. Affected trees may be attacked by Pestalotia, which causes a distinct brownish spotting on leaflets; when thus affected cadang-cadang disease has often been suspected. Unless attacked by leaf-spotting fungi leaves of deficiency-affected trees do not show blotching or spotting.

Nut and flower production is reduced and even absent in trees growing in the worst-affected areas.

In old cogon grass areas the yellowing generally is intensified. A mass of fallen, browned, dead leaves hanging around the trunk below the living leaves is characteristic. The presence of cogon grass is known to enhance deficiency troubles, through a definite lack of nitrogen. Trees growing in cogon grass areas, therefore, are in the same category as trees apparently suffering from some nutrient deficiency in cogon-free areas. In cogon grass areas young trees may be severely stunted.

Generally, deficiency troubles occur in blocks, varying in size according to the extent of the poor growing conditions, with all the trees showing a general lack of vitality. No scattered individually affected trees are common, as may happen with the infectious cadang-cadang disease. If observed from an airplane the characteristic aspect is a block or area of distinctly yellowed trees.

The trouble does not spread gradually as with an infectious disease, but is confined to spots or areas.

In those areas observed where older trees were manifesting possible deficiency, the early growth apparently was normal, but trees 35 to 40 years old showed a material reduction in yield.

Reduction in Yield

It is evident that yields in some of the older plantings, 30 to 40 years old, that had been highly productive, are falling off. In certain instances this reduction has been drastic. The

following examples, in plantations that we inspected in Quezon Province, will give an idea of what has happened. In one 50-year-old grove, the normal harvest in 1946 was 40,000 nuts per harvest; in 1950 it was 9,000 nuts per harvest. This was a typical case of gradual reduction in a grove not subjected to cogon grass. Many other groves like this were visited.

In another grove, 45 to 50 years old, that had been in cogon grass for three years, the normal harvest was formerly 10,000 nuts per harvest, at present it is 1,000 nuts per harvest.

A 35- to 40-year-old grove had produced 60,000 nuts per harvest before the war; after the war the harvest was 2,000 nuts, except the last harvest which was 10,000 nuts. Before the war the grove was in excellent condition, without cogon grass, but during and after the war it had been infested by cogon grass. The grove was plowed in January and May 1950, under the direction of Mr. Murray, and much of the plowed area was planted to legumes.

Discussion

Although experiment is needed to prove it, the cause of this trouble appears to be a deficiency of soil nutrients. The inhibitory effect that grass exerts on the activity of nitrifying soil organisms is well known; a definite deficiency of nitrogen results under such conditions and the grass also uses up a large part of the available soil nitrogen, thereby robbing orchard trees. The same condition undoubtedly exists in coconut groves infested by cogon grass.

In areas where cogon grass is not a problem the reduction in vitality and nut production might be due to some of the following conditions: general depletion of soils, presence of hard-pan, poor drainage, high water-table, growing of coconuts on poor, exhausted, or otherwise unfavorable soil types.

It is significant that little experimental data exist on soil fertility requirements, soil surveys, and general soil management for the main export crop of the Philippine Islands. It is important that long term experiments, covering at least 10 to 20 years' time, be started to determine the needs of coconuts for maximum production.

Detailed soil surveys of typical coconut areas should be made throughout the Philippines. Such surveys should include data on physical soil type of both top-soil and sub-soil, pH, and chemical analyses of essential nutrients. It might be well to start such surveys in areas noted for deficiency, and for comparison, in areas in which older coconuts are still producing excellent crops.

Intensive long-time replicated commercial fertilizer tests should be located in all different coconut soil type areas, planned to cover a long period of years and to prove whether or not yields can be increased and maintained in coconuts of different ages up to at least 50 to 70 years. It may be that production in the Philippines could be maintained or even increased with utilization of less acreage by increasing the yield on the best coconut soil types and eliminating coconuts from marginal lands.

The effect of plowing and cultivation prior to possible planting of legumes to kill all grass needs study.

Cover crop experiments should be enlarged. Much is already known about legumes, but growers need training in their use. Some excellent studies are now underway at the San Pablo Experimental plots and elsewhere. Legume cover crop tests should be extended to all different soil types in coconut areas and be so conducted as to determine the best types for killing cogon grass as well as for maintaining soil fertility.

It is important in all of these investigations that detailed exact data on the economic production of coconuts be obtained.

Literature Cited

1. Calinisan, Melanio R. The Tambolilid coconut. Plant Ind. Digest 11 (7): 16-19. 1948.
2. Celino, Martin S. A preliminary report on a blight disease (cadang-cadang) of coconut in San Miguel Estate, Albay Province. Philippine Jour. Agr. 13: 31-35. 1947.
3. _____ Progress report on experimental transmission of cadang-cadang disease of coconut. Philippine Jour. Agr. 13: 109-111. 1947.
4. _____ The cadang-cadang disease of coconut in the Philippines. Plant Ind. Digest 10 (1): 7-12. 1947.
5. Menon, K. P. V., and U. K. Nair. The wilt (root disease) of coconut in Travancore and and Cochin. Indian Coconut Jour. 3 (1): 5-10, 40-44. 1949. (Literature cited).
6. Ocfemia, G. O. The probable nature of cadang-cadang disease of coconut. Philippine Agriculturist 26: 338-340. 1937.

MANILA, PHILIPPINE ISLANDS

NEMATODE ROOT KNOT OF CINCHONA IN THE WESTERN HEMISPHERE[1]

Michael Szkolnik

Nematodes pathogenic to cinchona tree roots have been reported from several cinchona-growing areas of the eastern hemisphere but not from the western hemisphere (2, 3). The root knot nematode, Heterodera marioni (Cornu) Goodey, infests roots of several cinchona species in India and Java. Tylenchorhynchus alatus (Cobb) Filipjev infests roots of Cinchona succirubra in Africa. C. succirubra also is host for Tylenchus coffeae Zimm. in Java. Dorylaimus sp., together with H. marioni, has been isolated from cinchona seedlings.

Nematodes pathogenic to cinchona roots were discovered in two cinchona plantations in Guatemala: one, Finca El Naranjo, is at approximately 4000 ft. elevation on the southwestern slope of Volcano Santa Clara, while the other, Finca Moca, is at an elevation of approximately 3000 ft. on the southwestern slope of Volcano Atitlan.

Small, unthrifty trees of C. micrantha were observed in a five-year-old experimental planting at Finca Moca in October 1949. The dwarfed trees were scarcely three or four feet high while apparently healthy trees were about eight feet high. Branches were sparse and leaves were few and undersized on the stunted trees. Foliage on some stunted trees was pale green to yellow and red and abscised easily. Roots were examined on more than 25 unthrifty trees and root knots were numerous on 75 to 100 percent of the roots of all individuals. The enlargements often were twice the diameter of normal roots and in severe cases practically no functioning feed-roots remained. Living nematodes of both sexes were abundant in the root knot tissue that was examined microscopically. Ova in many different stages of development were numerous. Root specimens were sent to A. C. Tarjan of the United States Department of Agriculture, Division of Nematology, at Beltsville, Maryland and the nematode was identified as Meloidogyne incognita (Kofoid & White) Chitwood.[4]

Several undersized plants of Cinchona calisaya hybrid were growing in rows adjacent to the infested trees of C. micrantha. Roots of the hybrid were examined and although somewhat sparse -- which might account for the poor plant growth -- they formed a network of apparently normal rootlets, of normal color and uniform thickness. There were no nodular enlargements or other root knot symptoms. No examination was made for ectoparasitic nematoes.

Certain terraces at Finca El Naranjo have been used for approximately ten years for growing cinchona nursery stock. The nematode was discovered there in May 1950, in two-year-old stock of Cinchona succirbura side-grafted with hybrid cinchona clones of high alkaloid content. A random stunting of plants was practically the only evidence of nematode infestation, and detection was difficult without root examination. Foliage coloration was not a reliable symptom for locating infected plants. Roots were examined on 150 plants and the majority suffered relatively light infestation of part of the root system. An appreciable number suffered severe infestation of nearly the entire root system. The root knots often were two to three times the diameter of the normal root and contained large numbers of live nematodes. Some of the females were distended with ova and enlarged four to five times their normal diameter. Ova were in several stages of development. The nematode is very similar to and perhaps identical with the Meloidogyne incognita found on Cinchona micrantha.

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[1]Observations made while the author was controlling cinchona diseases for Experimental Plantations Inc., Guatemala, C. A., a subsidiary of Merck and Company Inc., Rahway, New Jersey.

[2]Koorders, S. H. Resultaten van een voorloopig mikroskopisch onderzoek eener wortelziekte van jonge kinaplantjes veroorsaakt door Heterodera aaltjex en een schimmel. De Cultuurgids 7: 901-919. 1906.

[3]Lombard, F. F. Review of literature on cinchona diseases, injuries, and fungi. U. S. Department of Agriculture, Bibliographical Bul. 9. 1947.

[4]Chitwood, B. G. Root-knot nematodes -- Part I. A revision of the genus Meloidogyne Goeldi, 1887. Proc. Helminth. Soc. Wash. 16: 50-104. 1949.

PHYTOPHTHORA ROOT ROT OF SAFFLOWER IN NEBRASKA CAUSED BY PHYTOPHTHORA DRECHSLERI[1]

Donald C. Erwin

Root rot of safflower (Carthamus tinctorius L.) has been observed since 1947 in western Nebraska (1). In a recent study at this station the causal organism was found to be Phytophthora drechsleri Tucker[2]. A high percentage of infection was readily obtained in greenhouse pathogenicity tests. Symptoms[3] may be obtained on safflower plants at all stages of growth from pre-emergence to near maturity. On young succulent plants a damping-off type of disease occurs. The appearance of necrotic lesions on the roots is followed by water-soaking and collapse of the cortical tissue of the lower stem which becomes shriveled, and the plant falls over and wilts. On older plants these symptoms on the lower stem do not occur; however, black, necrotic lesions encompass the roots and extend one or two inches above the ground on the lower stem. Symptoms on the roots and lower stems are soon followed by wilting of the entire plant, which often dies five to ten days after wilting. Often the leaves of older plants turn yellow before wilting.

In experiments in which soil temperature tanks were used, the greatest percentage of plants killed by root rot occurred at soil temperatures of 25° and 30°C, while only a small percentage were killed at 20° and none at 17°C. The optimum temperature for radial growth of the pathogen in culture was 30°C., which correlates closely with the optimum temperatures for development of disease. High soil moisture (above the moisture equivalent) was found to be conducive to rapid development of the root rot.

Some varieties have been reported to be resistant to root rot (2). No other control is known. Since the disease occurs at high temperatures, early planting and the limitation of irrigation to periods when the soil temperature is at a minimum, preferably 15° to 17° C., or lower, might be effective (3).

A more detailed report of this study will be published later.

Literature Cited

1. Claassen, Carl E., M. L. Schuster, and W. W. Ray. New diseases observed in Nebraska on safflower. Plant Dis. Reptr. 33: 73-74. 1949.
2. Claassen, Carl E., and Albert Hoffman. Safflower production in the western part of the northern great plains. Nebr. Agr. Exp. Sta. Circ. 87 (Revised). 1950.
3. Braun, A. J. Phytophthora root rot of guayule. Phytopath. 34: 933. 1944.

NEBRASKA AGRICULTURAL EXPERIMENT STATION, LINCOLN

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[1]Published with the approval of the Director as Paper No. 506, Journal Series, Nebraska Agricultural Experiment Station.
[2]The writer is indebted to Dr. C. M. Tucker for the identification of the organism.
[3]Similar symptoms have been observed on safflower in California, Arizona, and at Beltsville, Maryland, according to information obtained from Dr. C. A. Thomas of the United States Department of Agriculture, Division of Tobacco, Medicinal, and Special Crops.

FIELD DUSTING WITH DDT TO CONTROL THRIPS AND SPOTTED WILT IN TOMATOES

A. E. Michelbacher, M. W. Gardner, W. W. Middlekauff, and A. J. Walz

Because DDT used as a spray to control the thrips vectors of the spotted wilt virus showed promise under greenhouse conditions and in one field test (1), dusting with DDT was tested in 1946, 1947 and 1949 in tomato fields near Mt. Eden, California, a district known to be subject to spotted wilt infection.

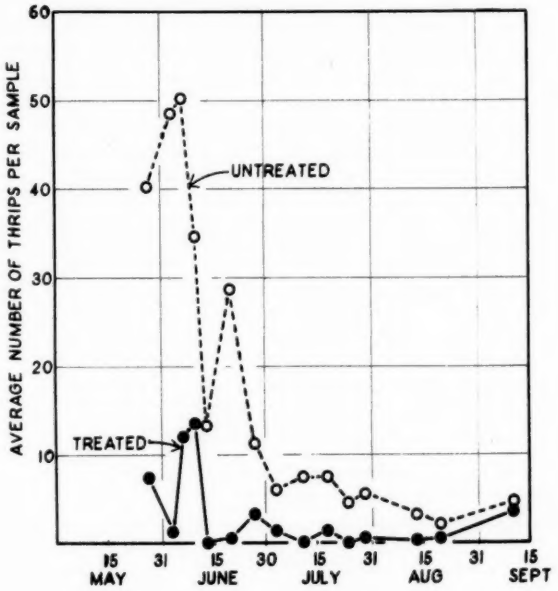

Figure 1. Reduction of thrips population in tomato field by dusting with DDT in 1946. Dust applied 5-16, 5-28, 6-11, 7-4, 7-19, and 8-1.

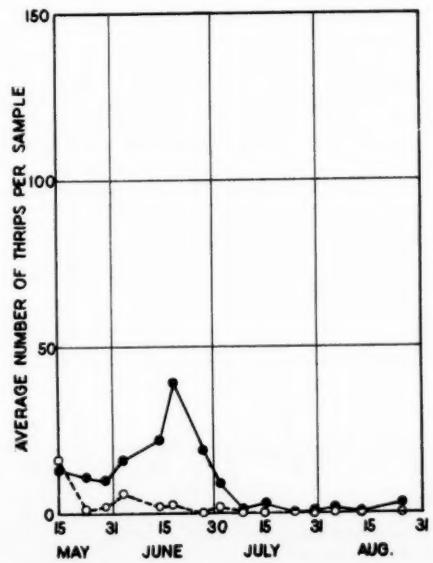

Figure 2. Reduction of thrips population in tomato field by dusting with DDT in 1947. Dust applied 5-17, 5-30, 6-9, 6-19, 7-3, 7-19.

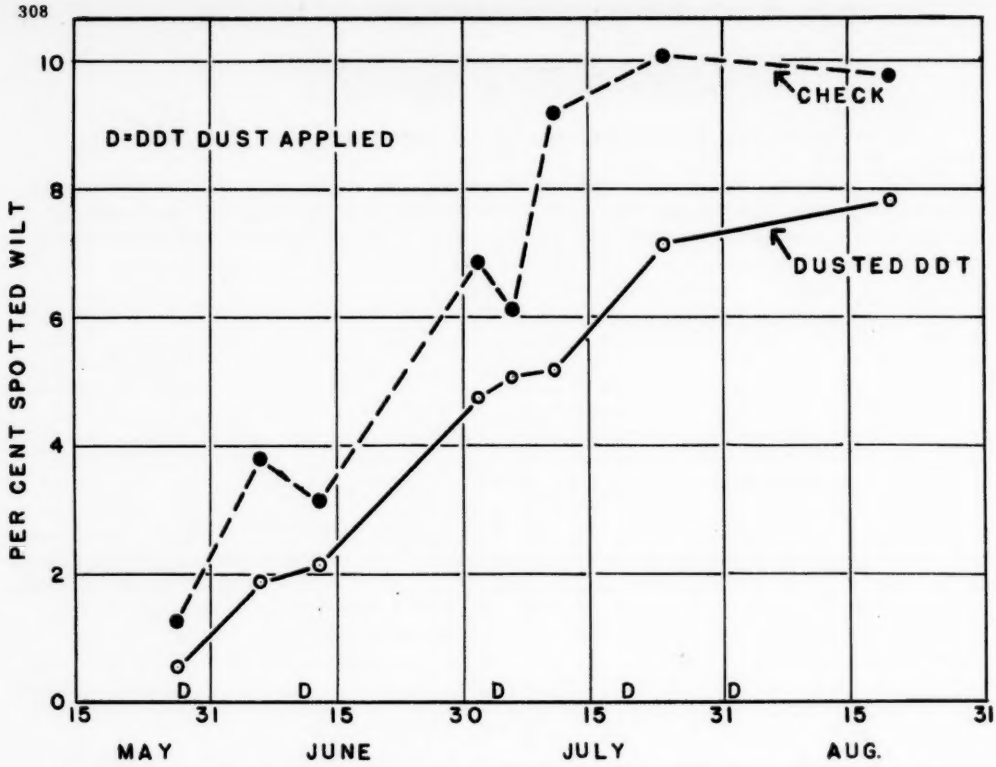

Figure 3. Reduction of spotted wilt infection in tomato field by dusting with DDT in 1946. Not proportional to the good control of thrips shown in Fig. 1.

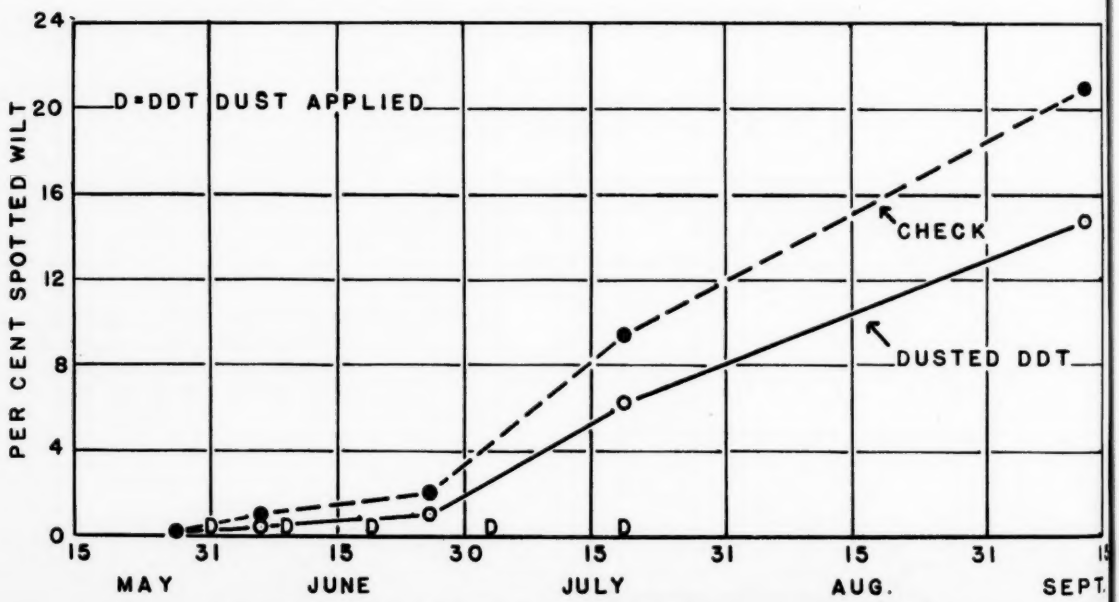

Figure 4. Reduction of spotted wilt infection in tomato field by dusting with DDT in 1947. Not proportional to the good control of thrips shown in Fig. 2.

Plots in two tomato fields were dusted 6 times in 1946. In one field there was considerable spotted wilt infection. The plots in this field were dusted with 5% DDT on May 16 and 28, June 11, July 4 and 19, and August 1. A good control of the thrips population was obtained (Fig. 1), as determined by counting the thrips on comparable samples of 10 to 25 leaves with a Schuh-Gray pea aphid separator (2). Frequently no thrips whatever were found in the samples from the dusted plots for several days after dusting.

The percentage of spotted wilt infection was determined by counts made at 8 intervals during the season (Fig. 3). While there was slightly less infection in the dusted plots, e.g., on July 24, 7 percent vs. 10 percent, the reduction in spotted wilt was by no means proportional to the reduction in thrips population.

Similar tests were made in 2 fields in 1947. The plots were dusted on May 17 and May 30 with 5% DDT and on June 9, 19, July 3 and 19 with 10% DDT + 25% sulfur to control the tomato russet mite. Frequent counts showed that an excellent control of thrips was obtained (Fig. 2). Again the reduction in percentage of spotted wilt was slight, e.g., 15 percent vs. 21 percent on September 12 in one field (Fig. 4).

In 1949 similar tests were repeated with a 5% DDT dust. The first application was late (June 29) and the control of thrips was not as satisfactory as in the other two years. Again there was only a slight reduction in the percentage of spotted wilt.

The results in the first two years when the reduction of spotted wilt was not at all proportional to the reduction in thrips population may be explained by assuming that there was little or no spread of infection from tomato to tomato within the field and that all of the infection resulted from flights or migration of infective thrips into the field from outside reservoirs of infection, such as ornamentals, as Bald (3) observed in Australia.

Literature Cited

1. Gardner, M. W., and A. E. Michelbacher. Controlling thrips and tomato spotted wilt with DDT. California Agr. Exp. Sta. Cir. 365: 35-38. 1946.
2. Gray, K. W., and Joe Schuh. A method and contrivance for sampling pea aphid populations. Jour. Econ. Ent. 34: 411-415. 1941.
3. Bald, J. G. Investigations on "spotted wilt" of tomatoes. III. Infection in field plots. Aust. Coun. Sci. Ind. Res. Bul. 106. 32 p. 1937.

DIVISIONS OF ENTOMOLOGY AND PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY

EFFECT OF FUNGICIDAL DRENCHES ON INCIDENCE OF LETTUCE DOWNY MILDEW IN THE SEED BED[1]

Frank A. Haasis and D. E. Ellis

Lettuce downy mildew, caused by Bremia lactucae Reg., resulted in serious loss of plants in isolated propagation frames in the vicinity of Burgaw and St. Helena, North Carolina, during

Table 1. Effect of soil fumigation and soil drench treatments with fungicides on incidence of lettuce downy mildew in the seed bed. Data are expressed as sin - $\sqrt{x}$ derived from infection readings in per cent[a].

Seed bed treatment[b c]				Disease Index (means of 3 replicates)		
Soil fumigation		Soil drench		Asgrow Great Lakes (Iceberg)	Asgrow Dark Green Cos (Romaine)	Average of both varieties
Material	Rate	Material	Rate / sq. yd.			
Methyl bromide	4 lbs. / 100 sq. ft.	Fermate 2-100	1/2 gal.	2.61	9.13	5.87
do	do	--	--	47.70	73.57	60.64
Chloropicrin	3cc. / sq. ft.	Fermate 2-100	1/2 gal.	4.23	3.68	3.95
do	do	--	--	65.80	78.04	71.92
do	do	--	--	51.48	66.71	59.09
--	--	Fermate 2-100	1/2 gal.	2.45	5.21	3.83
--	--	Tersan 2-100	do	6.00	4.91	5.45
--	--	Dithane Z-78 2-100	do	0.00	0.00	0.00
--	--	None	--	38.05	53.43	45.74
			Average	24.26	32.74	

L. S. D. 's	.05	.01
Two treatments (overall)	17.44	24.03
Two varieties (overall)	7.04	16.25
Two treatments on same variety	11.41	15.71
Two varieties on same treatment	13.64	27.28

[a]Special thanks are due Dr. R. J. Monroe, Institute of Statistics, N. C. State College, Raleigh, for the statistical treatment of the data.

[b]Notes relative to soil fumigation and soil drench treatments: Methyl bromide confined with Kraft paper and applied 11/8/49. Chloropicrin confined with water seal and applied 11/10/49. The second application of chloropicrin, in the one instance indicated, 11/12/49. Prior to injecting chloropicrin the second time the soil was carefully spaded thus replacing the surface layer with that from the subsurface zone having received near maximum fumigation from the first application.

Approximate soil temperature during fumigation 63°F.

First drench treatment applied 11/28/49 and continued at weekly intervals to 1/9/50.

[c]Indebtedness to the following chemical manufacturers is acknowledged:

Dow Chemical Company for methyl bromide (MC-2).

Innis-Speiden Company for chloropicrin (Larvacide).

E. I. duPont de Nemours and Company for Fermate (76% ferric dimethyldithiocarbamate) and Tersan (50% tetramethylthiuramdisulfide).

Rohm and Haas Company for Dithane Z-78 (65% zinc ethylene bisdithiocarbamate).

[d]Two applications, an interval of 2 days between first and second application.

[1]Contribution from Plant Pathology Laboratory, Raleigh and the Vegetable Research Laboratory, Wilmington of the North Carolina Agricultural Experiment Station and published with the approval of the Director as paper number 353 of the journal series.

January, 1950.

In the fall of 1949 an experiment was performed on a lettuce farm near Burgaw to determine the efficacy of two soil fumigants and three fungicides applied as drenches, in controlling damping-off of lettuce seedlings. Although damping-off was of little consequence in this test, downy mildew appeared in epiphytotic form between January 10 and 27. On January 17 an estimate was made of the amount of mildew infection appearing in the various treated plots, at which time it was found that plants treated with certain materials were free or relatively free of infection whereas others were heavily infected. On January 27 ten plants taken at random from five stations in each of three replicates planted with Iceberg lettuce and Romaine lettuce were examined for mildew. The results of the examination are given in Table 1, together with pertinent data relative to materials and methods employed, quantities applied, and application schedule.

Infection readings were high for the control plots and likewise for the plots that were fumigated. In marked contrast, however, plots that received drench applications of Fermate or Tersan contained but few infected plants, and plots that received drench treatments of Dithane Z-78 were free of infection.

The first drench treatments were applied on November 28, 1949, at the time of seeding, and were continued at weekly intervals until January 9, 1950; hence, a total of seven applications were made. Each drenched plot received approximately 4.5 grams of fungicide per square yard of soil surface per application, and a total of approximately 31.5 grams per square yard of soil surface for the season.

It should be noted, particularly, that the interval lapse between final drench application and final recording of data amounted to 18 days and that during this interval the lettuce plants made more than a onefold growth increase, thus reducing the possibility that mildew control accrued from a protective coating of the fungicides as a result of direct drench application to the plants themselves. Theoretically, therefore, complete mildew control with Dithane Z-78, and near complete control with either Fermate or Tersan, may have resulted from volatilization of the fungicides from the soil surface and condensation on the leaves on the one hand, or from plant absorption, either through roots, cuticle, or stomata, of the compounds directly or some fungicidal fraction of these compounds on the other, thus imparting a therapeutic effect. The data at hand are insufficient for logical development of possible explanations of the factual results.

The strain of the Romaine variety used is more susceptible to downy mildew than the Great Lakes strain, judged on the basis of inoculation and infection under natural conditions. Significance of the treatment x variety interaction appears to result from a different degree of treatment effect on the varieties since there are no significant reversals of treatment effect on the two varieties.

The results of this study re-emphasize the importance of applying fungicidal drenches to lettuce seedlings propagated in the cold frame.

NORTH CAROLINA AGRICULTURAL EXPERIMENT STATION

A PRELIMINARY REPORT OF RESULTS FROM SOME FUNGICIDE SPRAY TRIALS FOR CONTROL OF HELMINTHOSPORIUM LEAF BLIGHT OF SWEET CORN

Warren N. Stoner

Sweet corn, as a winter and spring vegetable crop in the State of Florida, has been increasing in importance. Relatively favorable prices and demand during the last two or three years have been the factors causing a large expansion of the acreage of this crop. If early estimates for the 1950-1951 growing season are realized the largest crop of sweet corn ever planted in this area will be produced. Growing of sweet corn on a large scale in Florida has been attended by an increasing number of problems. From a plant pathological standpoint leaf stripe, Helminthosporium turcicum has become a serious danger, and perhaps the limiting factor, to the continued production of this crop. Because of this threat, fungicide trials were started at the Everglades Experiment Station during the past season.

The disease can be found in the area throughout the year. Comparatively frequent rain, high humidity, and nightly heavy dews experienced locally favor the development of this disease. These climatic factors impose some restrictions on field trials. Observations of other workers in the area indicate that sprays are generally more effective than dusts. Consequently spray application was used for the investigations. A relatively short spray interval was used so that new growth could be protected as soon as possible, and the schedule was varied to replace materials washed off by rains. The experiment was conducted late in the spring and early summer of 1950, on a 6 x 6 latin square. The plots were situated on mature peat land, and each plot was 15 feet long with 5 rows 32 inches apart. The planting interval was 10 inches. Twelve foot alleyways were left between treatments and replicates. Standard disc nozzles were used and the materials were applied with trident headed hand wand using 250-300 pounds pressure. A sufficient amount of spray was used to obtain good coverage. The materials were not applied until the disease was apparent on the lower leaves of the plants. At this time the plants were 18 inches tall. An application was made every three days, if field conditions permitted, except that an extra spray was applied within 24 hours after each rain. The three day schedule was then resumed. Spraying was discontinued when the plants had tasseled. Under the above conditions it was necessary to make a total of eleven applications. The following materials were used at the amount per 100 gallons of water noted: Bioquin I (copper-8 quinolinolate 100 percent active material) 1 pound; Karathane W P - 25 (Dinitro capryl phenyl crotonate 22.5 percent, other nitrogen derivatives chiefly dinitro capryl phenol 2.5 percent. inert ingredients 75.0 percent) 1 1/2 pounds; Dry Parzate (zinc ethylene bisdithiocarbamate 65 percent, inert ingredients 35 percent) 2 pounds; S R - 406 (N - trichloromethylthio-tetrahydropntalimide 50 percent, inert ingredients 50 percent) 4 pounds of wettable powder; Caloclor (mercurous chloride 65 percent, mercuric chloride 30 percent, inert ingredients 10 percent) 2/3 pound plus Cadminate (cadmium succinate 60 percent, inert ingredients 40 percent) 1/6 pound. The check plots were treated the same as all other plots except no fungicide of any kind was applied to them.

The plots were rated twice by inspection, once when spraying was terminated, and again at the time of harvest. In each case the placement of each of the materials was consistent in all six replicates. The plots treated with Dry Parzate were best. Those sprayed with S R - 406 were second best. Bioquin I and Karathane W P - 25 were about equal and were placed third. The Caloclor Cadminate mixture was fourth. Repeated application of this mixture at the concentration used caused severe burning. The checks at each time of rating were rated the poorest.

The ears from each plot were harvested and weighed. With a few exceptions individual plot weights could be correlated with the inspection ratings. If the average yield weight of the six check plots is taken as unity, the average yield weight of the six plots treated with each material can be compared as follows: Dry Parzate 3.76, S R - 406 3.16, Bioquin I 2.29, Karathane W P - 25 2.2, Caloclor 1.46.

The husked ears from each plot in each replication were visually compared, and there was a definite difference between treatments. The checks had only a few grains on a few of the ears. There was a good filling of grain on the ears taken from the plots treated with Dry Parzate and S R - 406. Filling was poor in the ears taken from the Bioquin I plots and the Karathane W P - 25 plots. Ears from the Caloclor treatments were not much better than the checks. These findings correlate with the inspection rating and the average yield weight ratings.

Consistent differences, of effectiveness of the materials used in this investigation, are shown in the above ratings. These differences are large enough in the case of the two best materials, Dry Parzate and S R - 406, to offer hope that these materials can be used to control Helminthosporium turcicum on sweet corn. It should be emphasized that the control was obtained on an ex-

experimental basis, and further investigation will be necessary before these materials can be recommended for commercial use. Investigations on a larger scale are now beginning and a more detailed report on the experiment reported herein, and future findings, will be published at a later date.

EVERGLADES EXPERIMENT STATION, BELLE GLADE, FLORIDA

SEED TREATMENT TESTS ON OATS AND BARLEY IN 1950

R. W. Leukel

Nine fungicidal materials were tested as seed treatments for barley and oats in the spring and summer of 1950. These tests were similar to those of 1949[1] but were less extensive, as only two seed lots were treated and plantings were made at only four stations. In addition to the tests at Beltsville, Maryland, plantings were made at St. Paul, Minnesota, by M. B. Moore; at Fargo, North Dakota, by W. E. Brentzel; and at Pullman, Washington, by C. S. Holton. These workers also took the data at their respective stations, and their generous help is gratefully acknowledged.

Smut-infected Canadian oats were sent from Ames, Iowa, by H. C. Murphy, and stripe-infected Atlas barley from Davis, California, by Coit Suneson. Both of these contributions are gratefully appreciated.

The following materials were tested with regard to their effect on germination and their effectiveness in controlling the loose and covered smuts of oats and the stripe-disease of barley:

Ceresan M -- 7.7 percent ethyl mercury p-toluene sulfonanilide applied both as a dust and a slurry.

Aagrano 48 -- 1.8 percent ethoxy propyl mercury bromide.

Aagrano 250 -- 3.6 percent ethoxy propyl mercury bromide.

Semenon -- 8 percent isopropyl methyl mercuric acetate. (This was thought to be a 2-percent product and hence the seed was over-treated).

New Improved Ceresan -- 5 percent ethyl mercury phosphate.

Table 1. Percent emergence from treated seed of Canadian oats at 18°C. after storage periods of 1 and 6 months at room temperature; and percent of smutted plants grown from this seed in field plots at four stations in 1950.

No.	Seed treatment: Fungicide	Ounces per bu.	Percent emergence after storage for: 1 month	6 months	Percent infection in plots planted at: Beltsville, Md. March 30	Beltsville, Md. April 11	Fargo, N. Dak.	St. Paul, Minn.	Pullman, Wash.
1	Ceresan M	0.5	98	99	0	0	0	0	0
2	Aagrano 48	1.0	99	95	0	0	0	0	0
3	do	2.0	98	90	0	0	0	0	0
4	Aagrano 250	0.5	98	95	0	0	0	0	0
5	do	1.0	97	90	0	0	0	0	0
6	do	1.5	95	88	0	0	0	0	0
7	Semenon[a]	2.0	88	85	0	0	0	0	0
8	do	3.0	67	80	0	0	0	0	0
9	N. I. Ceresan	0.5	98	94	0	0	0	0	0
10	Untreated	---	98	90	12.8	22.9	13.6	45.7	14.7
11	Mercuran A.S.	0.5	97	95	0	0	0	0	0
12	do	1.0	96	94	0	0	0	0	0
13	Parson's Seed Saver	1.0	96	90	11.7	19.7	9.9	22.0	10
14	Mercuran A.L.[b]	0.25	97	90	.1	.5	1.7	.7	1.3
15	do[b]	0.5	93	88	.5	.9	1.7	1.7	2
16	Ceresan M slurry	0.5	94	99	.5	.2	0	1.0	0
17	Panogen[b]	0.75	97	95	1.1	.4	1.1	2.3	0
18	do[b]	1.00	97	96	0	0	0	0	0
19	do[b]	1.25	97	96	0	0	0	0	0
20	Untreated	----	98	90	13.2	23.1	15.7	40.3	11.4

[a] This 8 percent product was not labeled as such and hence seed was overtreated.

[b] Applied in liquid form by the "quick wet method."

[1] Leukel, R. W. Cooperative tests of seed treatments on spring grains in 1949. Plant Dis. Reptr. 33: 370-378. 1949.

Table 2. Percent emergence from treated seed of Atlas barley at 18°C., after storage for 1 and 6 months at room temperature; and the percentage of stripe-disease in plants grown from this seed in field plots at Beltsville, Maryland, 1950.

No.	Seed treatment: Fungicide	Ounces per bu.	Percent emergence after storage for: 1 month	6 months	Percent striped plants from seed sown: March 30	April 11
1	Ceresan M	0.5	96	98	0	0
2	Aagrano 48	1.5	96	96	0	0
3	Aagrano 250	.75	95	97	0	0
4	Semenon[a]	2.5	75	95	0	0
5	N. I. Ceresan	0.5	97	99	0	0
6	Mercuran A. S.	.75	98	96	0.3	0
7	Untreated	---	98	93	30.0	20.0
8	Parson's Seed Saver	1.0	96	94	14.0	3.5
9	Mercuran A. L.[b]	0.5	96	95	0.3	0
10	Ceresan M Slurry	0.5	92	97	0	0
11	Panogen[b]	.75	95	99	0.1	0
12	do[b]	1.0	93	95	0	0
13	do[b]	1.25	93	95	0	0
14	Untreated	---	98	97	32.0	22.0

[a]This was an 8-percent product but was not labeled as such, hence the dosage was four times as great as intended.
[b]Applied in liquid form by the "quick wet method."

Mercuran A.S. -- 3.5 mercury in the form of methoxy ethyl mercuric acetate applied as a dust.

Mercuran A.L. -- 7 percent mercury in the form of methoxy methyl mercuric acetate in soluble form and to be applied by the "quick wet method."

Parson's Seed Saver Dust -- a complex quarternary ammonium compound containing 3.8 percent mercury.

Panogen -- 2.1 percent methyl mercury dicyan diamide, a liquid to be applied by the "quick wet" method.

The methods used in treating, storing, germinating, and sowing the seed, and in taking data on the amount of disease present were about the same as described in 1949 (see footnote 1). Two plantings were made at Beltsville and one at each of the other stations. Sufficient oat smut developed in the check rows at all the stations to furnish a fairly adequate test for the fungicides in the control of this disease. These data are presented in Table 1. Only at Beltsville, however, were there enough stripe-infected barley plants in the check rows to furnish significant results on the control of stripe-disease. These data are presented in Table 2.

Emergence was not adversely affected by any of the fungicides except Semenon, which, because of insufficient labeling regarding its concentration, was applied at four times the intended dosage. Storage of the treated seed lots at room temperature for six months did not impair their viability.

The Ceresan dusts, which were used as standards of comparison, eliminated both the oat smuts (Ustilago spp.) and stripe disease (Helminthosporium gramineum). The Aagrano treatments, Semenon, and the two heavier applications of Panogen were equally effective. Ceresan M slurry did not eliminate oat smut completely, probably because of the shortcomings of applying a slurry treatment on a small scale. Mercuran A.S. (dust) eliminated oat smut and all but a trace of stripe disease. Mercuran A.L. was less effective in oat smut control. Parson's Seed Saver was relatively ineffective. The fact that Panogen, applied at 3/4 ounce per bushel, failed to eliminate oat smut and barley stripe completely, seems to confirm the writer's previous minimum recommended rate of one fluid ounce per bushel for this fungicide.

DIVISION OF CEREAL CROPS AND DISEASES, UNITED STATES DEPARTMENT OF AGRICULTURE

STEM RUST ON WHEAT AND OATS IN THE DAKOTAS AND MINNESOTA

(Abstracted by the Survey from reports by Donald G. Fletcher, Executive Secretary of the Conference for the Prevention of Grain Rust)

August 25 -- Black stem rust (Puccinia graminis) became epidemic on wheat and oats throughout the eastern half of the Dakotas and western Minnesota during the past three weeks. More than 300 fields of small grain were examined during a trip just completed through this territory.

Durum wheat of all varieties, including red durum (D5), is completely susceptible to at least one of the races of stem rust present this year. I have never seen a more virulent type of rust infection on any wheat -- Marquis in the 1920's and Ceres in 1935 not excluded -- than is now present in the fields of the spring-wheat area.

One of the more than 200 races of stem rust attacking wheat is designated as 15B. It is the most virulent race known to be present in North America. Race 15 was first identified in the early '20's. Race 15B, its close relative but much more virulent biotype, was not identified until about ten years ago. Before 1948, only an occasional collection of these races was made and in every case they were closely associated with rusted barberry bushes. In 1948 the number and distribution of race 15 increased, but in 1949 it almost disappeared. This year both races have been identified from rust collections made in our southwestern winter-wheat States as well as near barberries in the East.

Because of the fact that the rust now most prevalent in our northern wheat fields attacks such heretofore rust-resistant varieties as Stewart, Carleton, Nugget, and red durums, and also Mida, Redman, Lee, and Cadet bread wheats, it is believed that race 15B is widespread and here in quantity.

There is little question that this vicious race of rust is now independent of the barberry bush and will enter the wind-blown migration of rust from north to south and south to north in coming years. Susceptible grasses, such as wild barley, and volunteer grains between the Dakotas and Minnesota and southern Texas or northern Mexico will provide favorable stop-over spots on the southern journey of the rust this fall. Next spring the journey northward with the wind will start again. All conditions favorable for infection, growth, and distribution, must be present during the journey in both directions to produce a destructive rust epidemic next year in the spring-wheat area. This is true in spite of the fact that 15B has entered the picture.

The fact that the bread wheats this year are escaping with relatively little stem-rust damage has led many people to think that bread wheat is more resistant to 15B than the durums. This is not true. For example, Mida wheat, which on the average matures six days earlier than Stewart, shows just as much susceptibility if rusted plants at the same stage of maturity are compared.

Loss to bread wheat from stem rust alone should not exceed 2 or 3 percent in North Dakota. Loss in the other States of the spring-wheat area is believed to be even less. The chief reason for such a low loss this year is that the grain matured just ahead of the rust.

Loss to oats from stem rust and crown rust (P. coronata) combined, while varying considerably should not exceed 5 percent for the entire area. The oats actually escaped severe loss in most cases because they ripened before the rust could do its worst damage. The stem-rust and crown-rust situation has become more complicated because of the widespread occurrence of both race 7 and race 8 of stem rust and race 45 of crown rust. No commercial varieties are resistant to all three of these rusts. Most of the oats now grown are resistant to one of the important races and several minor races.

The durum-wheat loss caused by stem rust this year is very severe. In considering average damage, I am dividing the heaviest durum-producing area into three regions (shown on the map, Figure 1, but the loss indicated on the map is from the report of September 5). Durums in South Dakota, while heavily rusted in many cases at harvest time, are believed to have escaped severe injury. Southeastern North Dakota, outside the Red River Valley, also escaped with little damage. The valley proper above Fargo, both on the Minnesota and on the North Dakota side, should be included with the area designated as No. 1.

Area No. I -- Estimated average loss, 30 to 35 percent in bushels and one or two grades. Most grain within a week or ten days of harvest unless rain causes delay.

Area No. II -- Estimated average loss, 15 percent in bushels and one grade in 50 percent of production. Most grain ten days or more from harvest. Rust in this area did not develop as in Area No. I. Stems hardened, and cool weather prevented general extreme rusting expected earlier.

Area No. III -- including all durum west of Nos. I and II. Estimated average loss less than 5 percent. No loss of grade from stem rust.

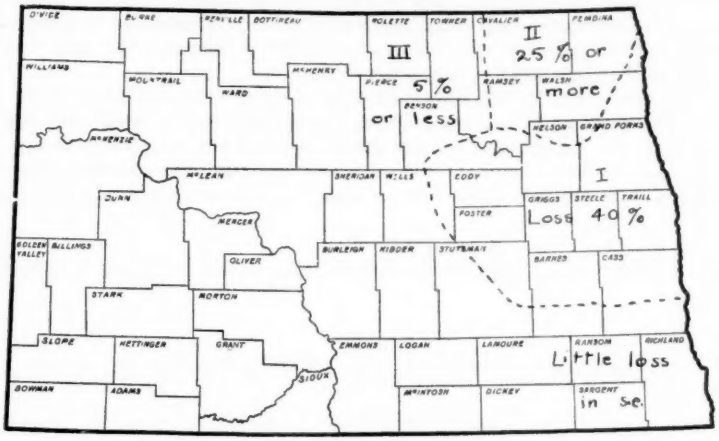

Figure 1. Preliminary estimates of probable loss caused by stem rust to durum wheat in North Dakota in 1950 (as of September 5)

It must be remembered that the stands of durum warranted an estimated average yield of 18 to 20 bushels. The loss of one-seventh to one-third of such a prospective yield will leave a large total production in bushels even if the quality is reduced.

In each of the first two areas mentioned there are regions of light rust damage and areas of very badly rusted durum. Stations may go one way or another, depending on local rainfall, date of seeding, development of rust, etc.

September 5 -- A four-day trip through the durum territory of North Dakota has just been completed.

Durum fields which had not reached mid-dough before the rust infection became severe are not maturing properly. The same fields that appeared to be ten days to two weeks from harvest on August 22 still seemed to be that far from the combine. Every area has its good, medium, and poor fields from a rust damage standpoint. Many of the individual fields are spotted and will thresh out both fairly plump and badly shriveled kernels. The resulting grain that comes to market will be quite mixed.

The earliest planted fields, with somewhat thin stands, are producing the best quality grain. Usually, the heaviest stands, planted on fallow or rich land, are producing the most rusted and shriveled grain. The early growth, when there was abundant moisture, went into the production of straw. When the soil became dry, the heavy, lush stands that were heavily infected with rust could not stand the high water requirements of the plants and the excess evaporation caused by the rust.

Rust damage to durum in Area I (see map, Figure 1) has increased to 40 percent. The average yield is believed to be about 10 bushels with serious loss of grade.

For Area II the rust on durums has materially increased. Late fields in an area from Devils Lake east to Lakota are good only for feed, and show a dirty, reddish-grey color, typical of severe rust infection. Many fields in the northern two-thirds of Area II are still two weeks plus from harvest and have not made appreciable progress since my last visit. The rust in the same period has increased from an average severity of less than 5 percent, to 25 percent at the present time. The necks of the plants, which are the most vulnerable portion, are generally infected. The final loss in Area II cannot safely be estimated now, as much of the northern two-thirds is still ten days to more than two weeks from harvest. Premature cutting may hasten the combining date. The loss of bushels certainly will be at least 25 percent, and if the late grain in the north does not fill for one or more reasons, the loss could be considerably higher. Lodging of a considerable acreage of heavy late grain around Agate and Rock Lake increases the probability of rust damage in those areas. Lodged grain furnishes ideal conditions for rust development. Storms in the north now would be very harmful.

Stem rust losses in Area III have increased slightly but I believe they will still average less than 5 percent. Only the later heavy fields will be injured materially. The large part of the durum acreage west of Cando and Devils Lake is out of danger from rust or is beating it to maturity by a narrow margin, with the result that much of it will be plump and of good color.

Oats have considerable stem and leaf rust infection but losses in most cases will be small. Good yields with satisfactory quality are being harvested.

Bread wheat is escaping with only a small amount of stem rust damage.

Considering the late start and the slow growing season in the Mid-west this year, our total production of small grain is remarkable. Although not a bumper crop, results are most satisfactory except in the case of durum wheat. On July 15 stem rust was present only in trace amounts in North Dakota. On the late durum crop it had time to increase and cause a severe epidemic.

CONFERENCE FOR THE PREVENTION OF GRAIN RUST, MINNEAPOLIS, MINNESOTA

DISEASES OF SMALL GRAINS IN ALABAMA

James A. Lyle

Small grains, particularly oats, are important winter crops in Alabama. However, diseases often limit the amount of grazing and seed harvested.

Surveys of grain fields and of small grain variety tests were made at intervals during the fall, winter, and spring of the 1949-1950 season.[1] Information obtained was supplemented by examination of specimens sent in by county agents and specialists in the Extension Service.

Additional information was obtained on the severity of barley diseases through experimental nurseries set up to determine the adaptability of certain strains of this crop in Alabama. These nurseries were located at the Plant Breeding Unit, Tallassee, and at the Black Belt Substation, Marion Junction. Two hundred and twenty-two different strains of barley, of spring and winter types, obtained from G. A. Wiebe of the Plant Industry Station, Beltsville, Maryland, were planted.

OATS

In general, oat diseases are much more prevalent in southern and central Alabama than in northern Alabama primarily because of more favorable weather conditons for the growth of the pathogenic organisms during the growing season of the host.

Helminthosporium blight (Helminthosporium victoriae), leaf blotch (H. avenae), and crown rust (Puccinia coronata) are the major oat diseases in Alabama. Black loose smut (Ustilago avenae), anthracnose (Colletotrichum graminicolum), halo blight (Pseudomonas coronafaciens), and mosaic (virus) were of minor importance.

No variety of oats, at any of the different locations in the State, showed good resistance to any of the major diseases.

Helminthosporium blight caused considerable damage to early planted fall oats in northern Alabama, especially on Victorgrain oats. In many instances farmers were compelled to replant. High humidity and warm temperatures, conditions favorable for the growth of the pathogen, prevailed during early fall.

In southern, central, and western Alabama, Helminthosporium blight caused more damage on plants approaching maturity than on plants in the seedling stage. Lodging and necrosis of lower nodes and root systems were evident among varieties affected in these areas. Splotching of leaves was particularly evident in central Alabama.

Helminthosporium leaf blotch (H. avenae) was much more prevalent during the 1949-1950 oat season than during the preceding two seasons. This disease was present to some extent on all varieties at all locations.

Crown rust was not so extensive and injurious on all varieties as in the preceding two seasons. Severe infestations of this disease occurred in Baldwin, Escambia, and Monroe Counties, which are located in southwestern Alabama. Every variety at each location showed light to heavy infection from this disease.

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[1] The Department of Agronomy and Soils conducts variety tests at 16 locations in the State.

Table 1. Disease ratings of oat varieties in Alabama, 1949-1950

Variety	Main Station Auburn			Black Belt Substation			Gulf Coast Substation			Wiregrass Substation		
	H.a.[a]	H.v.[b]	P.c.[c]	H.a.	H.v.	P.c.	H.a.	H.v.	P.c.	H.a.	H.v.	P.c.
Alabama 43-A	4	5	4	-	-	-	-	-	-	-	-	-
Anderson	5	5	3	4	5	3	5	5	4	4	5	4
Arlington (Ga.)	4	4	4	-	-	-	-	-	-	-	-	-
Arlington (N. C.)	4	4	3	4	4	3	4	5	4	3	3	4
Camellia	4	4	3	3	3	2	-	-	-	4	3	5
C. I. 5207	5	5	3	-	-	-	4	5	4	-	-	-
C. I. 5208	5	5	3	-	-	-	4	4	4	-	-	-
Coker 47-58	4	5	3	3	4	3	4	5	3	4	3	3
Delta Red	5	4	4	3	4	4	5	4	5	3	3	4
DeSoto	4	5	3	4	4	3	5	5	5	4	5	4
Fulgrain	5	5	3	4	4	3	4	5	4	4	4	3
Full Bright	5	5	3	3	5	3	4	5	4	4	4	3
Hastings 100 Bushel	4	4	4	4	4	4	4	4	5	3	3	4
Letoria	4	5	4	4	5	5	4	5	5	3	2	4
Nortex	4	4	3	3	4	4	4	4	5	3	3	4
Nortex 107	4	4	4	3	4	4	4	4	4	3	3	5
Red Rustproof 14	4	5	4	3	4	4	4	4	5	4	5	4
Stanton	5	5	3	4	4	2	5	4	4	4	5	4
Victorgrain BFS	5	5	3	4	5	3	5	5	4	3	5	4
Victorgrain 48-93	5	5	3	4	5	2	4	5	3	4	3	3

[a] Helminthosporium avenae
[b] Helminthosporium victoriae
[c] Puccinia coronata

Black loose smut occurred in widely scattered areas as localized infections in southern and central Alabama. It was most noticeable in Macon County in central Alabama.

Anthracnose was found at the Wiregrass Substation in southeastern Alabama; at the Black Belt Substation in western Alabama; at the Main Station, Auburn, in eastern Alabama; and at the Plant Breeding Unit, near Tallassee, in east-central Alabama. This disease was also isolated from a diseased oat sample sent in by a county agent from northern Alabama.

Halo blight and mosaic occurred on some varieties at all locations.

The oat varieties that were included in the 1949-1950 small grain variety test are listed in Table 1. The varieties Alabama 43-A and Arlington (Georgia) were included only in the test at the Main Station. C. I. 5207 and C. I. 5208 were included only at the Gulf Coast Substation, Fairhope, at the Main Station, and at the Plant Breeding Unit.

In Table 1 are given the disease ratings as recorded at the Main Station, Black Belt Substation, Gulf Coast Substation, and the Wiregrass Substation.

The disease ratings represent an average of four replications at each location. From a management and cultural standpoint, each location was handled in the same manner.

The varieties manifesting symptoms of Helminthosporium blight and Helminthosporium leaf blotch were rated by numbers 1 to 6, inclusive, depending on varietal susceptibility. These numbers indicated the following disease susceptibility:

1. Very resistant
2. Resistant
3. Slightly susceptible
4. Moderately susceptible
5. Susceptible
6. Very susceptible

Those varieties showing symptoms of leaf rust were rated by the modified Cobb rust scale.

In general, Helminthosporium blight (H. victoriae) was much more prevalent on the Victoria derivatives than on the Red Rustproof strains, while leaf rust (P. coronata) was much less prevalent on the Victoria derivatives than on the Red Rustproof strains (Table 1). This was true at

Table 2. Disease ratings of wheat varieties in Alabama, 1949-1950

Variety	Main Station Auburn			Black Belt Substation			Gulf Coast Substation			Wiregrass Substation		
	E.g.t.[a]	P.r.t.[b]	S.t.[c]	E.g.t.	P.r.t.	S.t.	E.g.t.	P.r.t.	S.t.	E.g.t.	P.r.t.	S.t.
Atlas 66	4	4	3	4	2	2	4	3	4	4	2	3
Chancellor	6	4	4	-	-	-	5	5	4	4	3	3
Coastal	5	3	4	4	2	3	4	3	4	5	2	3
Coker 47-23	4	2	3	3	2	3	4	3	4	5	4	1
Hardired	6	4	4	-	-	-	5	5	4	4	3	2
Redhart BFS	6	4	4	-	-	-	5	5	4	5	4	4
Sanford	6	4	4	-	-	-	5	5	4	5	3	3

[a] Erysiphe graminis tritici
[b] Puccinia rubigo-vera tritici
[c] Septoria tritici

all locations despite the fact that the disease infestation was not the same. Helminthosporium leaf blotch (H. avenae) varied in its disease incidence on all varieties at all locations (Table 1).

WHEAT

Wheat as a winter grain crop is of minor importance in this State.

Powdery mildew (Erysiphe graminis tritici), leaf rust (P. rubigo-vera tritici), and Septoria leaf blotch (Septoria tritici) were the most serious diseases affecting wheat in Alabama. Powdery mildew was the most prevalent and widespread of these three diseases. Septoria glume blotch (S. nodorum), loose smut (Ustilago tritici), and scab (Gibberella zeae) were of minor importance from a disease incidence standpoint.

The diseases on wheat were rated in a manner comparable to those on oats. In Table 2 is given the comparative susceptibility of the different varieties to the three most serious diseases, i.e., powdery mildew, leaf rust, and Septoria leaf blotch, at four different locations.

From the standpoint of disease incidence, the variety Coker 47-23 was by far the best at all locations (Table 2). Atlas 66 was almost as resistant to each disease at each location.

BARLEY

Very little barley is grown in this State. Root-rot (Helminthosporium sativum) and powdery mildew (Erysiphe graminis hordei) were the chief seedling diseases. Powdery mildew was the more prevalent. No barley line tested showed complete resistance to either of these two diseases.

Powdery mildew, leaf rust (Puccinia hordei), and spot blotch (H. sativum) were prevalent on most varieties after they passed the seedling stage. Loose smut (Ustilago nuda) occurred sporadically. Stripe (H. gramineum), net blotch (Pyrenophora teres), scab (Gibberella zeae), and Rhynchosporium scald (Rhynchosporium secalis) were of minor importance.

ALABAMA POLYTECHNIC INSTITUTE, AUBURN

DISEASES REDUCE VETCH SEED CROP IN TENNESSEE IN 1950

J. Lewis Allison, Homer D. Wells, and J. O. Andes

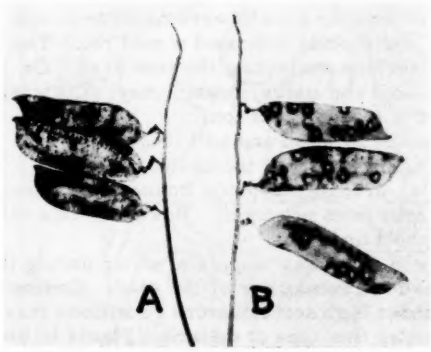

Figure 1. Pods of Vicia villosa showing symptoms of:

A. Colletotrichum villosum
B. Ascochyta pisi

In recent years large acreages of hairy vetch, Vicia villosa, have been grown for seed in Lawrence, Lincoln, Franklin, and adjacent Tennessee counties. The 1950 seed crop, however, was greatly reduced by disease damage.

A field survey made June 12 and 13, followed by microscopic and cultural studies of collected specimens, revealed that most of the damage was due to anthracnose (Colletotrichum villosum) and blight (Ascochyta pisi). Both pathogens were universally present and were causing severe losses in all fields. Leaves, stems, and seed pods were heavily infected, and C. villosum and A. pisi were readily isolated from all parts, including seeds. These pathogens can be expected to be widely disseminated in all seed harvested from this region this season.

Rapid vegetative growth of the vetch during the unusually warm winter months followed by a period of high rainfall during May and the first part of June contributed to the build up of the epiphytotics this year. Heavy growth of the vetch and its companion wheat crop resulted in severe lodging, thus forming a more favorable environment for the development and spread of both pathogens.

DIVISION OF FORAGE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, AND THE TENNESSEE AGRICULTURAL EXPERIMENT STATION, KNOXVILLE, TENNESSEE

SOME NEW RECORDS OF PLANT DISEASE OCCURRENCE

TYPHULA SPECIES PATHOGENIC ON MINT[1]

By A. Steenland and E. Burke

Peppermint plants exhibiting yellowed lower leaves and spindly growth were noted in Grand Island, Oregon in early March. Examination of the roots and stolons indicated a root rot. The diseased area appeared as a dark-brown, hardened outer surface encircling the root area. On examination the center proved to be browned tissue with a soft and watery consistency. Cultures from the rotted areas revealed a basidiomycete associated with this condition.

In April, healthy plants in the greenhouse were inoculated by stem and soil inoculation with sclerotia and mycelium of the above isolate. Reisolation again produced the basidiomycete.

This fungus has been identified as a species of Typhula, probably Typhula itoana Imai. Species identification has not been made, as no sporophores have been produced. However, this is the first known report of the pathogenic association of Typhula and mint.

The soil at Grand Island has a high moisture content and a constant supply of water during the winter and early spring months. Sprinkle irrigation is used the remainder of the year. Common association of Typhula species as pathogenic on grasses under high soil moisture conditions may indicate that the organism may be limited to mint grown under this type of culture. Plants in the stand were examined in June and this organism has affected the general vigor of the mint. To date, it has not been found in other mint stands in the State.

OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

NATURAL OCCURRENCE OF ASTER YELLOWS ? ON TRIFOLIUM HYBRIDUM

By Frank P. McWhorter

This records an unusual disease of Alsike clover observed by the grower, Mr. John Craven, in his second year field near Merrill, Oregon, during July 1950. Mr. Craven, who has grown Alsike clover for seed in Klamath county for many years, stated that he had not previously seen clover plants growing like those he had staked for our observation. The affected plants were erect, standing about 20 inches higher than normal plants, with a habit not clover-like, but typical of some member of the umbelliferae. The change in habit is an expression of the disease symptom "witches broom". At the points where normal inflorescences should arise, the stipules and (unit) peduncles were replaced by rosettes of peduncles bearing tiny inflorescences and leaves only 1/5 normal size. Also a few flowers formed in the axillary locations where the stipules should have been. Diseased plants formed no seeds, thus being a total loss. Fortunately only a few plants were infected in the entire field.

A very similar disease attributed to western aster yellows has been observed on alfalfa, onions, and many other plants in southeastern Oregon. This Merrill case is our first record of an aster yellows like disease on Alsike clover in Oregon.

No garden or weed plants were found in the vicinity that could be considered potential sources of the virus. Unfortunately plant tests to prove the presumed etiology cannot be arranged this season.

OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

RUGOSE MOSAIC AND UNMOTTLED CURLY DWARF ON POTATOES IN NEW HAMPSHIRE

By M. C. Richards

A rotobeater was used on the potato test plots in the fall of 1949 to kill the vines. This or later insect infestations on the remaining stalks was favorable for the spread of rugose mosaic in the Mohawk and B 76-43 U.S.D.A. varieties. On July 1, 1950, 60 percent of the Mohawk and 95% percent of the B 76-43 plants showed infection. A trace only was found in the other varieties. B 76-43 plants did not show the rugose symptom of the leaves, but the remaining symptoms of yellowing, death of the leaves, and the shoestring appearance of the dead leaves and petioles were produced.

About 1 percent of the plants of B 76-43 showed symptoms of unmottled curly dwarf (Folsom, Maine Agr. Exp. Sta. Bul. 446: 87-95, 1946). This is the first report of this disease in New Hampshire.

UNIVERSITY OF NEW HAMPSHIRE, DURHAM

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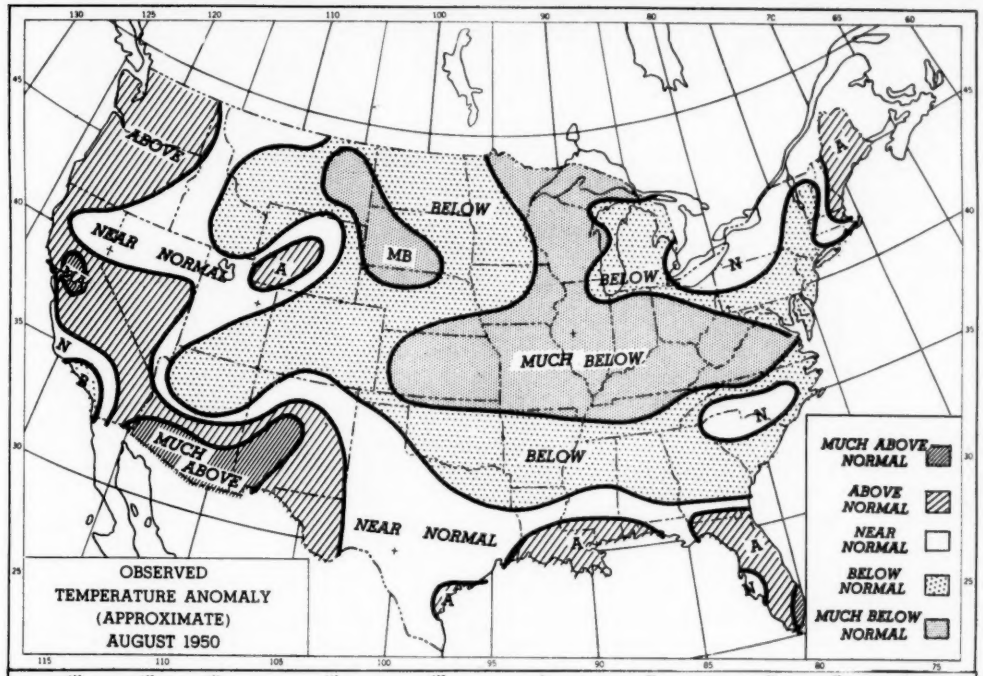
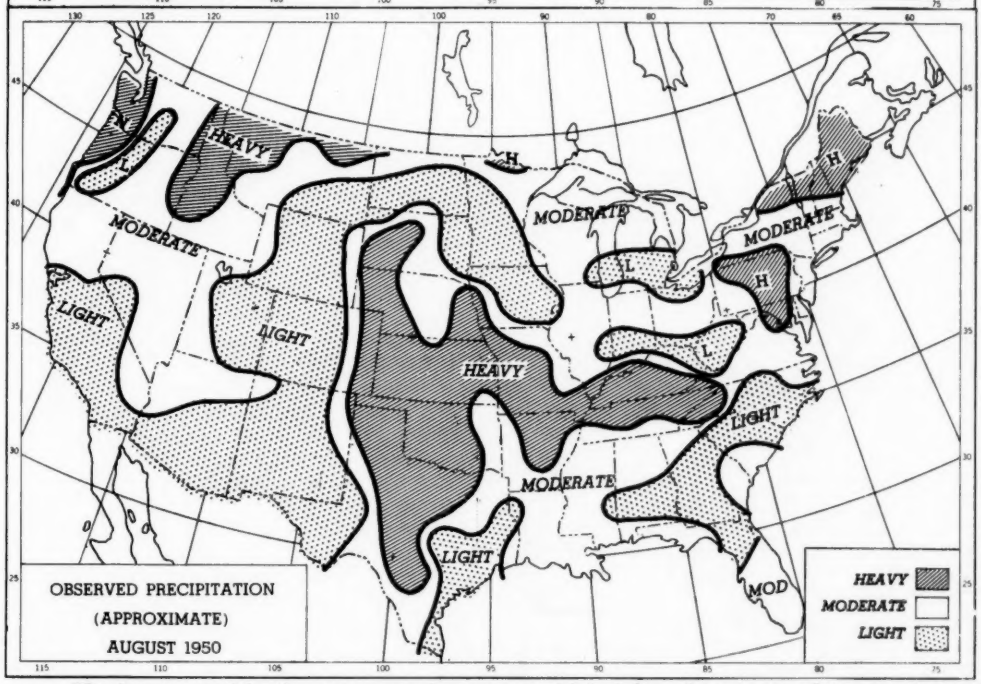
[1] Published as Technical Paper No. 643 with the approval of the Director of the Oregon Agricultural Experiment Station. Contribution of the Department of Botany and Plant Pathology.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

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